



Akt (phospho Tyr315) Monoclonal Antibody

Catalog No	BYmab-14540
Isotype	IgG
Reactivity	Human;Mouse;Rat
Applications	WB
Gene Name	AKT1/AKT2/AKT3
Protein Name	RAC-alpha serine/threonine-protein kinase/RAC-beta serine/threonine-protein kinase/RAC-gamma serine/threonine-protein kinase
Immunogen	The antiserum was produced against synthesized peptide derived from human AKT1/2/3 around the phosphorylation site of Tyr315/316/312. AA range:281-330
Specificity	Phospho-Akt (Y315) Monoclonal Antibody detects endogenous levels of Akt protein only when phosphorylated at Y315.
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source	Monoclonal, Mouse,IgG
Purification	The antibody was affinity-purified from mouse antiserum by affinity-chromatography using epitope-specific immunogen.
Dilution	WB 1:500-2000
Concentration	1 mg/ml
Purity	≥90%
Storage Stability	-20°C/1 year
Synonyms	AKT1; PKB; RAC; RAC-alpha serine/threonine-protein kinase; Protein kinase B; PKB; Protein kinase B alpha; PKB alpha; Proto-oncogene c-Akt; RAC-PK-alpha; AKT2; RAC-beta serine/threonine-protein kinase; Protein kinase Akt-2; Protein kinase B
Observed Band	56kD
Cell Pathway	Cytoplasm . Nucleus . Cell membrane . Nucleus after activation by integrin-linked protein kinase 1 (ILK1). Nuclear translocation is enhanced by interaction with TCL1A. Phosphorylation on Tyr-176 by TNK2 results in its localization to the cell membrane where it is targeted for further phosphorylations on Thr-308 and Ser-473 leading to its activation and the activated form translocates to the nucleus. Colocalizes with WDFY2 in intracellular vesicles (PubMed:16792529).
Tissue Specificity	Expressed in prostate cancer and levels increase from the normal to the malignant state (at protein level). Expressed in all human cell types so far analyzed. The Tyr-176 phosphorylated form shows a significant increase in expression in breast cancers during the progressive stages i.e. normal to hyperplasia (ADH), ductal carcinoma in situ (DCIS), invasive ductal carcinoma

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(IDC) and lymph node metastatic (LNMM) stages.

Function	catalytic activity:ATP + a protein = ADP + a phosphoprotein.,disease:Defects in AKT1 are associated with breast cancer (BC) [MIM:114480]. BC is an extremely common malignancy, affecting one in eight women during their lifetime.,disease:Defects in AKT1 are associated with colorectal cancer (CRC) [MIM:114500].,disease:Defects in AKT1 are associated with susceptibility to ovarian cancer [MIM:604370]; also called susceptibility to familial breast-ovarian cancer type 1 (BROVCA1).,domain:Binding of the PH domain to the phosphatidylinositol 3-kinase alpha (PI(3)K) results in its targeting to the plasma membrane.,domain:The AGC-kinase C-terminal mediates interaction with THEM4.,enzyme regulation:Three specific sites, one in the kinase domain (Thr-308) and the two other ones in the C-terminal regulatory region (Ser-473 and Tyr-474), need to be phosphorylated for its full activation.,function:Gene
Background	The serine-threonine protein kinase encoded by the AKT1 gene is catalytically inactive in serum-starved primary and immortalized fibroblasts. AKT1 and the related AKT2 are activated by platelet-derived growth factor. The activation is rapid and specific, and it is abrogated by mutations in the pleckstrin homology domain of AKT1. It was shown that the activation occurs through phosphatidylinositol 3-kinase. In the developing nervous system AKT is a critical mediator of growth factor-induced neuronal survival. Survival factors can suppress apoptosis in a transcription-independent manner by activating the serine/threonine kinase AKT1, which then phosphorylates and inactivates components of the apoptotic machinery. Mutations in this gene have been associated with the Proteus syndrome. Multiple alternatively spliced transcript variants have been found for this gene. [provided by RefSeq, Jul 2011]
matters needing attention	Avoid repeated freezing and thawing!
Usage suggestions	This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

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